To the Editor:

Stents implanted into hard, calcified lesions do not always fully expand, even though they are inflated at very high pressure, and once the device has been released the problem is difficult to solve. The ablation of the stent and the calcium that protruded through the stent rings by means of high-speed rotational atherectomy reduces wall thickness, facilitating balloon dilation. This technique, which is not without potential risks, has been successfully employed in three patients.1,2

A 70-year-old hypertensive, hypercholesterolemic man was admitted to our hospital for acute coronary syndrome with ST-segment depression in anterior leads. The results of physical examination, chest radiography, blood cell count and standard laboratory tests, including serial determinations of creatinine phosphokinase level and troponin T expression, were normal. In addition to the treatment he was receiving at the time of admission, consisting of atenolol, atorvastatin and aspirin, clopidogrel, and intravenous infusion of nitroglycerin was initiated and cardiac catheterization was performed. The left ventricle, which was hypertrophic and presented moderate hypokinesia of the apical region, preserved an ejection fraction of 63%. The bifurcation of the left main coronary artery presented a stenosis of 40% and subtotal occlusion of the ostium of the circumflex artery. This vessel, which was occluded distally, was being supplied via inadequate collateral circulation branching from the right coronary artery, which presented severe stenosis of the middle segment. All these lesions were successfully treated by means of stent placement. The left anterior descending coronary artery (Figure, A), possibly the culprit vessel, presented an extensive severe, calcified stenosis of the middle segment. After predilatation using a 2.5×25-mm Maverick balloon, a 2.75×24-mm Driver stent was implanted; however, a portion of the distal third did not expand completely, presenting a diabolo-like image (Figure, B). An attempt to post dilate using a 3.0×13-mm Powersail balloon inflated to a pressure of 25 atmospheres failed (Figure, C), and therefore rotational atherectomy using a 1.75-mm olive-shaped burr (Figure, D) was carried out. Afterwards, post dilatation resulted effective, and a 3.0×12-mm Taxus pharmacoactive stent was deployed into the previously implanted stent (Figure, E). The final angiographic result was excellent (Figure, F) and there were no adverse events. The postoperative course was satisfactory and the patient, when discharged from the hospital 48 hours later, was asymptomatic. At 5 months, coronary angiography revealed the absence of restenosis in the Taxus stent.

The efficacy of rotational atherectomy in situations of incomplete expansion of stents implanted in severely calcified lesions has been demonstrated in three previously reported cases in which there were no problems associated with the erosion of the metal.1,2 The success may be attributed to the ablation of the stent rings and the calcium that protruded through them, resulting in the thinning of the wall. Although

Figure. Angiographic sequence. The arrows indicate the location of the underexpanded ring in each image. A: anterior descending coronary artery prior to the interventional procedure. B: predilatation and implantation of a stent (2.75×24 mm), showing the underexpanded ring. C: unsuccessful postimplantation dilatation at 25 atmospheres. D: intra-stent rotablation with a 1.75-mm olive-shaped burr. E: repeated postimplantation dilatation followed by drug-eluting stent placement (3.0×12-mm Taxus). F: final satisfactory angiographic result.
certain potential complications, such as the distal embolization of metal particles or late thrombosis, could develop, they did not occur in any of the four cases documented in the literature. Our case confirms the feasibility of this technique in special cases.

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REFERENCES

Letters to the Editor

Unusual Electrocardiographic Presentation of Arrhythmogenic Right Ventricular Cardiomyopathy

To the Editor:

Arrhythmogenic right ventricular cardiomyopathy or dysplasia (ARVD) is a genetically determined heart muscle disease in which the myocardium is replaced by fibrofatty tissue. It is associated with arrhythmias, heart failure and sudden death.1 Typical symptoms include palpitations, dizziness and syncope. Sudden death is often the first sign, especially in young people, and it coincides with some type of strenuous physical activity; in some series, it accounts for up to 20% of all sudden deaths in young adults.2 With the introduction of automatic implantable defibrillators, many of these deaths can be prevented. However, the prevention of arrhythmic death requires timely diagnosis, which depends on the ability of the primary care physician and the specialist to be aware of and to diagnose this disease.

The diagnosis is established on the basis of certain major and minor criteria involving the evaluation of the family history, the depolarization or repolarization-induced electrocardiographic changes, arrhythmias, structural changes and ventricular dysfunction, and on the histopathological features.3 At the present time, there are a number of registries underway for the purpose of analyzing the validity of these criteria and broadening them, if appropriate.4 The typical clinical signs, together with T-wave inversion in leads V1 to V3 present in over 50% of the patients,5 or the appearance of the epsilon wave in 30%,6 raise the suspicion of this disease; however, there are reports of cases in which the electrocardiographic features were less common, but the risk of sudden death was the same.7 We present the case of a 31-year-old man, a professional soccer player, who was referred to our unit because of occasional palpitations. After initial evaluation and the verification of the absence of a family history of heart disease, we examined the electrocardiogram, which revealed negative T waves in leads II, III, and aVF and ventricular premature beats with right bundle branch block (RBBB) morphology (Figure 1). During 24-hour Holter monitoring, several episodes of nonsustained ventricular tachycardia (NSVT) were recorded, as were premature beats with RBBB morphology that appeared to originate in the left ventricular apex. Transthoracic echocardiography revealed a slightly dilated left ventricle with apical akinesia. Given these findings, the patient underwent single photon emission computed tomography (SPECT) to study myocardial work, reaching 20 MET without symptoms, but with frequent premature beats and self-limited episodes of NSVT similar to those recorded during 24-hour Holter monitoring. The scintigraphic images disclosed a fixed apical perfusion defect. In view of these findings, coronary arteriography was carried out, but no lesion of any type was observed; however, ventriculography revealed the presence of left ventricular apical akinesia. Right ventricle was dilated and unstructured, with akinetic/dyskinetic areas and diastolic septal bulge (“stack of coins”; Figure 2). As ARVD was suspected, magnetic resonance imaging was requested. It revealed fatty infiltration of the right ventricle. ARVD with involvement of the left ventricle was diagnosed.